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OALJ NO.: 1998-BLA-00855

BRB NO.: 01-0376 BLA

In the Matter of

JOSEPH E. MASSIE
Claimant

v.

CONSOLIDATION COAL COMPANY
Employer

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party-in-Interest

Appearances:

Frederick K. Muth, Esquire (Hensley, Muth, Garton & Hayes), Bluefield, West Virginia for the Claimant

Mary Rich Maloy, Esquire and Ann B. Rembrandt, Esquire (Jackson & Kelly), Charleston, West Virginia for the Employer

Before: Daniel F. Sutton
Administrative Law Judge

SECOND DECISION AND ORDER ON REMAND AWARDING BENEFITS

I. Statement of the Case

This matter, which arises from a claim for benefits filed on July 31, 1997 by Joseph E. Massie (Massie) against the Consolidation Coal Company (Consolidation) under Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended (the Act), 30 U.S.C. §901 *et seq.*, is before me a third time pursuant to a second remand from the Benefits Review Board (the Board). *Massie v. Consolidation Coal Co.*, BRB No. 01-0376 BLA (January 10, 2002)

(unpublished). The Act provides for the payment of benefits to coal miners who are totally disabled due to pneumoconiosis, and to the survivors of a coal miner whose death is due to pneumoconiosis. 30 U.S.C. §901(a).

The District Director, Office of Workers' Compensation Programs (OWCP) denied Massie's claim on December 29, 1997. Director's Exhibit "DX" 15. Massie requested a formal hearing which was conducted before me on October 21, 1998. At the hearing, the parties stipulated that the Massie had 23 years of coal mine employment and that the evidence was sufficient to establish total disability pursuant to 20 C.F.R. §718.204(c) (1997).[1] Consolidation contested whether Massie suffers from pneumoconiosis arising out of coal mine employment and whether his conceded total disability is due to pneumoconiosis. In my initial decision and order, I found that although the evidence was insufficient to establish pneumoconiosis pursuant to 20 C.F.R. §718.202(a)(1)-(3), Massie had proved the presence of pneumoconiosis by a preponderance of the medical opinion evidence pursuant to 20 C.F.R. §718.202(a)(4). In making this latter finding, I credited opinions from Drs. Rasmussen and Forehand over contrary opinions Consolidation introduced from Drs. Zaldivar, Jarboe, Fino, Loudon and Castle. I further found that Massie established that his pneumoconiosis arose out of his coal mine employment and that his total disability was due to pneumoconiosis. Based on my determination that he had established all elements of entitlement under the Act, I granted awarded Massie benefits to be paid by Consolidation.

Consolidation appealed to the Board, challenging my findings regarding the presence of pneumoconiosis and cause of Massie's total disability. Initially, the Board affirmed my findings that the evidence did not establish the presence of pneumoconiosis pursuant to 20 C.F.R. §718.202 (a)(1)-(3). *Massie v. Consolidation Coal Company*, BRB No. 99-0973 BLA (June 30, 2000) (unpublished), slip op. at 2 n.1. However, the Board agreed with Consolidation that I erred in not addressing whether Dr. Forehand's opinion was sufficiently reasoned, and it held that I failed to explain my basis for finding that Dr. Rasmussen's opinion was supported by that of Dr. Forehand, noting that Dr. Rasmussen diagnosed legal pneumoconiosis, while Dr. Forehand diagnosed clinical pneumoconiosis. Slip op. at 2-3. The Board also found that I improperly

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[1] The regulations implementing the Act were revised effective January 19, 2001. 65 Fed. Reg.79,920-80,107 (December 20, 2000). The provisions pertaining to total disability, which were previously set out at 20 C.F.R. §718.204(c), was recodified in the revised regulations to 20 C.F.R. §718.204(b). The mining industry challenged the revisions, but the United States District Court granted the Secretary of Labor's motion for summary judgment, denying all challenges to the new regulations. *Nat'l Mining Ass'n v. Chao*, 160 F. Supp.2d 47 (D.D.C. 2001). Thereafter, the Court of Appeals for the District of Columbia Circuit upheld most of the challenged regulations but ruled that certain regulations, none of which are relevant to consideration of this case on remand, could not be applied retroactively. *National Mining Ass'n. v. Dep't. of Labor*, 292 F.3d 849 (2002). All citations herein to the regulations will be to revisions effective on January 19, 2001, with the exception that references to the earlier decisions will include citations to the version of the regulations in effect at the time the decision was issued.

discredited the opinions of Drs. Zaldivar, Jarboe, Fino, Loudon, and Castle because they failed to identify an alternative etiology for Massie's lung disease. In this regard, the Board stated that a physician's opinion need not establish a definitive alternative etiology for a claimant's lung disease and is sufficient to rule out the presence of pneumoconiosis if it effectively rules out coal mine employment as a cause. Slip op. at 3, citing *Hall v. Director, OWCP*, 12 BLR 1-133 (1989) and *Casella v. Kaiser Steel Corp.*, 9 BLR 1-131 (1986). Thus, the Board vacated my finding that the medical opinion evidence is sufficient to establish the presence of pneumoconiosis pursuant to 20 C.F.R. §718.202(a)(4), and it remanded the case to me for further consideration with instructions that I weigh all of the evidence bearing on the existence of pneumoconiosis together, as required by *Island Creek Coal Co. v. Compton*, 211 F.3d 203, 210-11 (4th Cir. 2000), to determine whether Massie suffers from the disease. *S*lip op. at 3-4. Finally, the Board found merit in Consolidation's contention that I failed to make a separate finding regarding whether the evidence was sufficient to establish that Massie's total disability is due to pneumoconiosis pursuant to 20 C.F.R. §718.204(b) because I did not separately consider the etiology of the Claimant's disability. Accordingly, it instructed that if I determined on remand that Massie had established the existence of pneumoconiosis arising out of coal mine employment, I must consider whether Massie's total disability is due to pneumoconiosis pursuant to 20 C.F.R. §718.204(b). Slip op. at 4, citing *Robinson v. Pickands Mather & Co.*, 914 F.2d 35 (4th Cir. 1990).

On remand, I reconsidered the medical opinion evidence pursuant to the Board's instructions. I found Dr. Forehand's medical finding of pneumoconiosis to be unsupported by the objective medical evidence because it is based on a positive x-ray interpretation that is contrary to my finding that a preponderance of the x-ray evidence did not establish presence of pneumoconiosis. I further found that Dr. Forehand's unsupported diagnosis of medical pneumoconiosis based on his interpretation of the radiographic evidence neither supported nor contradicted Dr. Rasmussen's diagnosis of legal pneumoconiosis. Decision and Order on Remand at 3. I next reconsidered the medical opinions offered by Consolidation's experts and found them all to be deserving of diminished weight. I discounted Dr. Zaldivar's opinion because I found that it is based on an assumption, *i.e.*, that pneumoconiosis produces an obstructive rather than a restrictive impairment, that I found to be hostile to the Act's definition of pneumoconiosis. Decision and Order on Remand at 4, citing *Warth v. Southern Ohio Coal Co.*, 60 F.3d 173, 174-75 (4th Cir. 1995) (holding that chronic obstructive lung disease is encompassed within the statutory definition of pneumoconiosis and that the ALJ should have rejected as hostile to the Act an opinion that a miner's obstructive respiratory impairment was due exclusively to emphysema caused by smoking since it was based on a premise that coal dust inhalation causes a restrictive rather than a purely obstructive impairment), and I distinguished *Lane v. Union Carbide Corp.*, 105 F.3d 166, 175 (4th Cir. 1997), where the Court concluded that Dr. Zaldivar's statement that simple pneumoconiosis would "not be expected" to cause pulmonary impairment was not hostile to the Act because his analysis demonstrated that he did not rely on any hostile assumptions but rather based his opinion on the medical evidence and considered the possibility that the claimant's simple pneumoconiosis caused a totally disabling respiratory impairment. Decision and Order on Remand at 5. I also noted that Dr. Zaldivar's assumption regarding the ventilatory characteristics of pneumoconiosis is contradicted not only by Dr. Rasmussen, but by Dr. Jarboe, another of

Consolidation's experts, and that Drs. Rasmussen and Jarboe, unlike Dr. Zaldivar, both cited medical literature to support their opinions. As for Dr. Jarboe, I found his opinion that simple pneumoconiosis is "rarely, if ever" disabling to be in conflict with the Act under *Thorn v. Itmann Coal Co.*, 3 F.3d 713, 719 (4th Cir. 1993) (physician opinion that simple pneumoconiosis does not "as a rule" cause total disability is based on a premise antithetical to the Act). Decision and Order on Remand at 4-5. I next considered the opinion from Dr. Fino who concluded that the significant oxygen transfer abnormality shown in arterial blood gas studies conducted by Drs. Rasmussen in 1997 and Dr. Zaldivar in 1998 was inconsistent with a condition related to coal mine dust exposure because the abnormality was not present in 1994 when Massie's oxygen saturation was measured and because development of significant oxygen transfer abnormality within a couple of years after an individual leaves the mines is not consistent with a coal mine dust pulmonary condition. I found Dr. Fino's comparison of the 1994 measurements with the 1997 and 1998 blood gas studies problematic since the record reflects that the 1994 testing utilized a different methodology (oximetry) from that employed by Drs. Rasmussen and Zaldivar (drawing a sample of arterial blood which is the only method authorized by the Regulations), and involved a substantially lower level of exercise. Based on my review of the records, I determined that the level to which Massie was exercised in 1994 was markedly lower than the exercise levels achieved in 1997 and 1998, and I concluded that Dr. Fino's reliance on the "normal" 1994 testing to surmise that the Claimant's impairment developed too rapidly after 1994 to be consistent with a disease related to coal mine dust inhalation is not well-reasoned. Decision and Order on Remand at 6. With regard to the opinion from Dr. Loudon, I noted that he used the medical or clinical definition of pneumoconiosis instead of the broader legal definition set forth in the Act and Regulations, and I rejected his finding of no pneumoconiosis for the following reasons:

> Significantly, Dr. Loudon concedes that he was unable to identify the cause(s) of the Claimant's gas exchange abnormalities, and he stated that the type of abnormality seen in the Claimant is common to several conditions (*e.g.* COPD, interstitial lung disease and asthma) which can fall within the legal definition of pneumoconiosis if related to or aggravated by coal mine dust exposure. And, while he did suggest that the Claimant's gas exchange abnormalities are the likely result of either an increase in the alveolar-arterial gradient for oxygen and carbon monoxide or from a ventilation-perfusion mismatch, he did not state that these conditions are not related to or aggravated by the Claimant's extensive occupational exposure to coal mine dust. Therefore, I find that Dr. Loudon's opinions carry little probative force in rebutting Dr. Rasmussen's well-reasoned diagnosis of legal pneumoconiosis.

Decision and Order on Remand at 8. Lastly, I reconsidered Dr. Castle's opinion and found that he failed to provide any rationale to support his conclusion that Massie most likely suffers from interstitial pulmonary disease that is unrelated to his exposure to dust in coal mine employment. Decision and Order on Remand at 8-9. On the other hand, I credited Dr. Rasmussen's diagnosis of legal pneumoconiosis as well-reasoned and supported by the objective medical evidence, and I found that Massie had established the existence of pneumoconiosis under 20 C.F.R.

§718.202(a)(4). Decision and Order on Remand at 9. I then weighed all of the relevant evidence together as ordered by the Board, and bearing in mind the distinction between clinical and legal pneumoconiosis, I found that the inconclusive x-ray evidence did not directly contradict or offset the medical opinion evidence which I had found sufficient to establish the existence of legal pneumoconiosis. Accordingly, I concluded that Massie had established by a preponderance of the evidence that he suffers from pneumoconiosis as defined in the Act. Decision and Order on Remand at 10. Finally, I concluded that Massie's pneumoconiosis arose out of coal mine employment and that his totally disabling pulmonary impairment is due to pneumoconiosis, and I again awarded him benefits to be paid by Consolidation. Decision and Order on Remand at 10-11.

Consolidation once again appealed to the Board which again reversed. *Massie v. Consolidation Coal Co.,* BRB No. 01-0376 BLA (January 10, 2002) (unpublished). Although the Board held that I had permissibly rejected Dr. Forehand's diagnosis of clinical pneumoconiosis as unreasoned, slip opinion at 3-4, it found several errors in my analysis of the opinions rendered by Consolidation's experts. First, the Board agreed with Consolidation that I exceeded my expertise by commenting on the level to which claimant exercised during the 1994 arterial blood gas study and the level to which claimant exercised during the 1997 and 1998 arterial blood gas studies and, thereby, improperly substituted my opinion for that of Dr. Fino. On this basis, The Board vacated my finding that the evidence is sufficient to establish the existence of pneumoconiosis at 20 C.F.R. §718.202(a)(4), and it remanded the case for further consideration of the evidence. Slip opinion at 5. The Board then proceeded to consider Consolidation's exception to my finding that Dr. Rasmussen's opinion is better reasoned than the opinions of Drs. Castle, Fino, Jarboe, Loudon and Zaldivar. In this regard, the Board noted that contrary to my statement that no physician had attributed Massie's lung impairment to cigarette smoking, the record reflects that Dr. Jarboe opined in a report dated August 12, 1998 that the most likely cause of claimant's mild airflow obstruction is cigarette smoking and not dust exposure (EX 8) and that Dr. Castle had testified at a deposition taken on October 16, 1998 that it is very likely that Massie's obstruction is related to his tobacco abuse (EX 16 at 22). The Board thus held that inasmuch as I had mischaracterized the medical opinion evidence, I erred in finding Dr. Rasmussen's opinion better reasoned and entitled to more weight than the contrary opinions of record. Slip opinion at 5. The Board further held that I had erroneously rejected Dr. Zaldivar's medical opinion as conflicting with *Warth* because Dr. Zaldivar did not assume that coal mine employment can never cause chronic obstructive pulmonary disease. Slip opinion at 6-7. The Board similarly held that I erred in discrediting Dr. Jarboe's opinion as hostile to the Act because Dr. Jarboe did not foreclose all possibility that simple pneumoconiosis can be disabling. Slip opinion at 7. The Board further instructed that in the event that I find the evidence sufficient to establish the existence of pneumoconiosis pursuant to 20 C.F.R. §718.202(a)(4), I must then weigh all types of relevant evidence together in accordance with *Compton* in determining whether Massie has established the existence of pneumoconiosis. Slip opinion at 7. Lastly, in view of its determination to vacate my finding that Massie had established the existence of pneumoconiosis, the Board vacated my findings that Massie's pneumoconiosis

arose out of his coal mine employment and that his total disability is due to pneumoconiosis. Slip opinion at 7.

Upon reconsideration of the relevant evidence in accordance with the Board's instructions, I conclude for the reasons discussed below that the Massie has met his burden of establishing that he suffers from pneumoconiosis as defined in the Act, that his pneumoconiosis arises from his coal mine employment, and that he is totally disabled due to pneumoconiosis. Accordingly, I again award him benefits to be paid by Consolidation.

II. Findings of Fact and Conclusions of Law On Remand

A. Reconsideration of the Medical Opinion Evidence Pursuant to 20 C.F.R. §718.202(a)(4)

In view of the Board's finding that I had mischaracterized the medical evidence in my earlier decision, I will again summarize the pertinent medical evidence of record, albeit in greater detail than previously.[2]

Dr. Rasmussen

Dr. Rasmussen conducted a medical history and examination at the OWCP's request on August 29, 1997. DX 10. He noted that Massie had worked for 24 years in coal mining and had no smoking history. Id. at 1-2. He concluded that Massie suffers from severe pulmonary insufficiency as evidenced by marked hypoxia with exercise, and he attributed this condition to coal mine dust exposure, stating that "[t]he only known risk factor for this patient's severe pulmonary insufficiency is his coal mine dust exposure." *Id.* at 4.

Dr. Rasmussen was examined regarding his medical findings at a deposition taken on October 13, 1998. CX 3. He testified that Massie's test results indicated to him that Massie is suffering from a fairly severe interstitial type chronic lung disease, of which one variety "would clearly be coal worker's pneumoconiosis." *Id.* at 6. Dr. Rasmussen stated that he had no reason to suspect any diagnosis other than pneumoconiosis, such as diffuse interstitial pulmonary fibrosis or collagen vascular disease because he had no symptoms of physical findings suggestive of these disease processes. *Id.* at 7. Dr. Rasmussen was questioned about Dr. Zaldivar's report and diagnosis of idiopathic diffuse interstitial pulmonary fibrosis, and he testified that one would need to examine a specimen of lung tissue in order to distinguish this condition from coal worker's pneumoconiosis, and he added that coal mine dust exposure itself can produce diffuse interstitial pulmonary fibrosis. *Id.* at 8. He explained that a study of coal miners in Wales and southern West Virginia showed that coal miners have a 17 to 18 percent incidence rate for diffuse interstitial pulmonary fibrosis, as compared to a four percent rate for the general population, and

[2] Dr. Forehand's reports are not included in the summary based on the Board's approval of my rejection of his opinions as unreasoned.

he stated that other studies have elucidated the cellular and biochemical mechanisms by which coal mine dust exposure can produce diffuse interstitial pulmonary fibrosis. *Id.* at 9-10. Dr. Rasmussen testified that coal mine dust exposure can produce obstructive lung disease, but he added that he has seen hundreds of miners with no significant airway obstruction but a significant gas exchange impairment independent of x-ray findings and the usual physical findings associated with interstitial fibrosis. *Id.* at 10. He was also questioned about a study he had done involving non-smoking coal miners, and he testified that while about 40 percent of the miners in the study had airway obstruction, some of the miners had a pattern of significant gas exchange impairment absent airway obstruction which is different from the usual pattern of impairment seen in cigarette smokers. *Id.* at 11. He described a significant gas exchange impairment without obstruction as fairly typical in coal miners, and he stated that this pattern, which is distinguishable from the pattern seen in smokers, is what he found in Massie's case. *Id.* at 12.

With regard to the Massie's cigarette smoking, Dr. Rasmussen was asked whether the 13 year history cited in Dr. Zaldivar's report would impact on his opinions, and he responded that it would not because he would expect an impairment related to cigarette smoking to be primarily obstructive in nature where Massie has very minimal or slight obstruction. *Id.* at 12-13. He was also asked to comment on the opinions from Drs. Loudon, Fino, Jarboe and Castle that Massie's impairment is not one normally seen in patients with coal worker's pneumoconiosis, to which he responded,

> I disagree quite heartily with their opinions having seen hundreds of coal miners with this exact pattern of impairment, and I don't believe that those hundreds of coal miners have all had diffused interstitial pulmonary fibrosis; in fact, many of them at autopsy have an element of fibrosis as well as an element of emphysema along with coal worker's pneumoconiosis, but I would disagree that it is not – that this pattern is not typical of coal miners' lung disease.

Id. at 13-14. Dr. Rasmussen testified that his x-ray findings were insufficient to justify a diagnosis of pneumoconiosis; however, he added that he has seen Massie's pattern of impairment in a lot of coal miners with little or no abnormality seen on x-ray, and he stated that it is possible to have debilitating *legal*, as distinguished from *medical*, pneumoconiosis absent x-ray findings. *Id.* at 14-15. He explained the difference between a diagnosis of *medical* or *clinical* pneumoconiosis and *legal* pneumoconiosis as follows:

> I don't think you could actually diagnose coalworkers' pneumoconiosis based on that absent x-ray findings. In other words, clinically you make a diagnosis of pneumoconiosis based on an occupational history and a positive x-ray or a piece of lung tissue showing the anatomical lesion. On the other hand, I would still be in a position to say I believe that, for example, this Mr. Massie has coalworkers' pneumoconiosis based on his work history and his impairment in function, although I can say – what I'll say is I can say that his coal mine dust exposure is

> responsible for his impaired respiratory function absent significant x-ray abnormalities.

Id. at 16. In rendering a diagnosis of legal pneumoconiosis in the absence of x-ray findings of clinical pneumoconiosis, Dr. Rasmussen testified that he considers factors such whether there is a known medical history to suggest another cause of interstitial lung disease, whether there is a significant history of exposure to coal mine dust, and whether the pattern of impairment is typical of many coal miners. *Id.* at 16-17.

Dr. Rasmussen was further questioned about the significance of Massie's normal diffusing capacity test in 1994 and his abnormal diffusing capacity results in later testing conducted in 1997 and 1998. He responded that diffusing capacity can vary based on factors such as status of rest, and he stated that "it is also possible for the disease processes caused by coal mine dust exposure to progress even following termination of exposure." *Id.* at 17-18. In support of his statement that a condition related to coal mine dust exposure can continue to progress or worsen following termination of exposure, Dr. Rasmussen referred to a study which, he said, showed that the alveolitis process that occurs in individuals exposed to coal mine dust persists in some cases more than five years after the individual leaves the coal mines. *Id.* at 18. He concluded that Massie suffers from pneumoconiosis and that it is a major factor contributing to his totally disabling respiratory impairment. *Id.* at 19.

On cross-examination, Dr. Rasmussen acknowledged that he sees individuals in his practice with diffuse interstitial fibrosis where he is unable to identify the cause, and he explained that in cases where there is no occupational history that would put the individual at risk, he would conduct further testing such as looking for rheumatoid factors or anti-nuclear antibodies and having a lung biopsy performed. *Id.* at 22-23. He explained that he did not believe that further testing was medically indicated in Massie's case to because he presented "such a classic picture of coalworkers' pneumoconiosis or coal mine dust induced lung disease . . . ." *Id.* at 26, 29-30. He also acknowledged that he has seen cases where miners have 30 to 40 years of coal mine exposure with no measurable impairment despite significant x-ray abnormalities, and he stated that the vast majority of miners whom he has seen over the years have no impairment. *Id.* at 24.

On re-direct examination, Dr. Rasmussen testified that he believed that it was far more likely that Massie's interstitial pulmonary fibrosis is caused by his coal mine dust exposure than being classified as idiopathic because his pattern is typical of a coal mine related impairment. *Id.* at 31-32. He agreed with Dr. Zaldivar's statement that idiopathic fibrosis damages the capillary beds in the lungs, but he added that coal worker's pneumoconiosis also damages capillary beds. Finally, Dr. Rasmussen testified that Massie's gas exchange abnormality is caused by destruction of lung tissue, either due to centrilobular emphysema or diffuse interstitial pulmonary fibrosis, both of which can be caused by coal mine dust exposure and both of which destroy capillary beds. *Id.* at 32-33.

Dr. Rasmussen is board-certified in internal medicine but not in the sub-specialty of pulmonary disease because he never took the qualifying examination. *Id.* at 5. In the 35 years that he has been practicing medicine in West Virginia, he has specialized in the study of miner's disease, and he has held positions as the chief medical office of the Appalachian Coal Miners Research Unit of the U.S. Public Health Service Division of Occupational Health, chief of the pulmonary section of the Appalachian Regional Hospital and medical director of the Appalachian Pulmonary Laboratory. *Id.* at 4. He testified that he has examined approximately 40,000 coal miners, and he has published six or seven articles. *Id.* at 5.

Dr. Zaldivar

Dr. Zaldivar examined Massie on May 6, 1998 and found the same pattern of pulmonary abnormality that Dr. Rasmussen detected during his examination. EX 4. However, he concluded that the pattern of impairment is indicative of interstitial lung disease instead of coal worker's pneumoconiosis which, he said, "produces an obstructive and not a restrictive nor [sic] interstitial impairment." *Id.* at 2. Dr. Zaldivar speculated that the Claimant's pulmonary abnormalities are caused by "either interstitial pulmonary fibrosis which will require biopsy to establish the cause, or loss of vascular beds by chronic pulmonary embolism or vasculitis." *Id.* at 3.

Dr. Zaldivar testified regarding his medical opinions at a deposition taken on October 12, 1998. EX 15. He described the x-ray findings diagnostic of coal worker's pneumoconiosis and testified that he would expect to see an obstructive impairment as well as blood gas and diffusion abnormalities if the impairment were severe enough. *Id.* at 7-8. He further testified that there are five types of
emphysema – (1) centrilobular which he described as emphysema for smokers, (2) senile emphysema which mimics centrilobular, (3) panlobular emphysema, (4) paraseptal emphysema and (5) focal emphysema which he described as the emphysema of coal worker's pneumoconiosis. *Id.* at 9-10. He stated that Massie has two risk factors for lung disease, smoking and coal mine employment, and he stated that 30 years of coal mine employment is sufficient to cause lung disease. *Id.* at 10-11. He also stated that the Claimant's smoking history of 12 or 13 years is significant enough to cause an airway obstruction in a susceptible individual. *Id.* at 12. Dr. Zaldivar also pointed out that Massie is overweight at 64 inches and 256 pounds, and he stated that this is "important" because obesity can cause a restriction by decreasing capacity within the thorax. *Id.* at 13-14. He added that he had interpreted Massie's chest x-ray as evidencing possible enervation the right diaphragm which he described as a "congenital problem in this case aggravated by being overweight" which "[s]ometimes . . . causes the diaphragm to malfunction." *Id.* at 15-16. However, he later retracted this testimony when Consolidation's attorney pointed out to him that there is a discrepancy between his examination report which lists Massie's weight as 256 and Dr. Rasmussen's report which gives Massie's weight as 153:

> I see the problem now. I am sorry. I did mention two hundred fifty-six pounds. I believe that this was a mistake because I see the breathing test, my breathing test, is a hundred and fifty-six pounds. So I have to take back what I said regarding a

> normal body weight would cause the oxygen uptake to be higher. And also, when I mentioned it in the examination, it should be a hundred and fifty-six pounds. So weight doesn't enter into this at all. His weight is about right for him. Everything else is the same, but the weight is incorrect. It was a typing error in the physical examination which I carried through on my discussion today, although I did not make a point of it when I interpreted the blood gasses. The blood gasses were interpreted independent of the weight. So I'm sorry about that.

Id. at 50. Dr. Zaldivar agreed with Dr. Rasmussen's finding of a mild airway obstruction. *Id.* at 18-19. He stated that his diffusing capacity study produced low results which, in conjunction with normal ventilatory function studies, led him to think of pulmonary fibrosis as a cause of Massie's problem. *Id.* at 21-22. He testified that there are many causes of pulmonary fibrosis, but simple coal worker's pneumoconiosis is not one. *Id.* at 22. Dr. Zaldivar further testified that Massie's blood gas study during exercise showed a "real significant drop" which fit well with the results of his diffusing capacity study. *Id.* at 23-24. He stated that the test results led him to conclude that Massie has a lung disease but no cardiac problem. *Id.* at 25.

Dr. Zaldivar was asked to address the cause of Massie's drop in oxygen with exercise and his impairment in diffusing capacity, and he responded that, based on his experience and everything that he has read in the medical literature, the degree of diffusing capacity impairment seen in this case without an airway obstruction does not occur in coal worker's pneumoconiosis because there is no mechanism by which coal worker's pneumoconiosis can damage the capillary bed to the extent where the diffusing capacity is going to be decreased. *Id.* at 29-30. On the other hand, he stated that pulmonary fibrosis, 80 percent of which is idiopathic, and vasculitis caused by collagen vascular disease and lupus produce such an impairment. *Id.* at 30-31. He added that a study by Dr. Rasmussen in 1988 of coal miners given lung scans established no pattern which would link coal worker's pneumoconiosis and the type of impairment seen in this case. *Id.* at 32-33. While Dr. Zaldivar agreed that Massie's coal mine employment history is the only risk factor that can be identified for causing his respiratory impairment, he testified that he believes that Massie's impairment is not related to his coal mine employment and that one must analyze the data before identifying an etiology. *Id.* at 33. In his view, Massie "just happened to be a coal miner who has lung disease which is not related to his occupation." *Id.* at 34.

Dr. Zaldivar testified that his conclusion on the cause of Massie's impairment is not based "solely" on his negative x-ray interpretation, and he stated that if Massie were his patient, he would recommend a high resolution CT scan and lung biopsy "because I would tell him that there is really no diagnosis until a piece of tissue is obtained to try to identify the cause of the diffusion abnormality which is not coal worker's pneumoconiosis." *Id.* at 34, 36. He further testified that Massie does not have a case of lung impairment induced by cigarette smoking because smoking-related ventilatory obstruction and emphysema would not explain Massie's low diffusing capacity and abnormal blood gasses with exercise. *Id.* at 37-38. At the same time, he reiterated his belief that the patter of impairment seen in this case "does not fit what is published about coal worker's

pneumoconiosis." *Id.* at 39. Dr. Zaldivar also attempted to explain his view that coal worker's pneumoconiosis is the "opposite" of pulmonary fibrosis:

> Coal worker's pneumoconiosis, when it causes an impairment, it causes an airway obstruction which may progress to the point where it is crippling. Pulmonary fibrosis is shrinkage of lung tissue. It causes scarring in the lungs which, as it progresses – which at first begins with a low diffusing capacity. Then, as it progresses, it will cause the total lung capacity and vital capacity to be reduced as the lung shrinks as a result of scarring of some sort. That is what gives you the low diffusing capacity. The earlier manifestation is a low diffusion with a normal chest x-ray, in fact. Then, as it progresses, then the total lung capacity is reduced and vital capacity is reduced. But that's at the opposite end of the spectrum of obstructive diseases which is what coal worker's pneumoconiosis causes. I'm talking about simple pneumoconiosis. Complicated pneumoconiosis is different. Complicated pneumoconiosis causes shrinkage of the lung tissue by destroying it.

Id. at 40-41. Dr. Zaldivar then added that the "normal" oxygen transfer results obtained by oximetry testing in 1994 supported his conclusion that the Claimant's impairment is unrelated to his occupation:

> If he had trouble with oxygen transfer as a result of his occupation, he would have had it in 1994 after he worked thirty years in the coal mines. He would not develop this in the subsequent two years after not being exposed to coal dust any longer two or four years later. He has no longer been exposed to dust. The chest x-ray has not shown any progression of the disease. There is nothing related to his occupation at this late date after finishing work [which would] cause this kind of problem. So that goes along with my diagnosis of a disease process totally unrelated to his occupation.

Id. at 41-42. He further stated that coal worker's pneumoconiosis can "occasionally" progress, provided that it is present radiographically, but "it is not reasonable to expect a development of pulmonary abnormalities afterwards when it wasn't present when he quit work and he had been exposed to all the dust that he was going to be exposed to in his lifetime." *Id.* at 42-43. He continued that he would not implicate Massie's occupation as a cause of his impairment even if his blood gasses had been abnormal in 1994 because Massie's pattern of low diffusing capacity in the face of essentially normal ventilatory studies is inconsistent with coal worker's pneumoconiosis. *Id.* at 43-44.

On cross-examination, Dr. Zaldivar confirmed his belief that coal worker's pneumoconiosis causes an obstructive ventilatory impairment. He also testified that the type of obstructive impairment caused by coal worker's pneumoconiosis is similar to the obstructive disease caused by cigarette smoking, and he stated that one would not be able to differentiate between the two based on breathing tests alone. *Id.* at 46. He stated that the discrepancy

between the 12 or 13 year smoking history he reported and the no smoking history reported by Drs. Rasmussen and Forehand would not in any way change his opinions. *Id.* at 47. Dr. Zaldivar testified that it is highly unlikely that a disabling type of pneumoconiosis would not be seen radiographically and that he based his opinion that Massie does not have coal worker's pneumoconiosis on the absence of an airway obstruction. *Id.* at 48. Finally, he testified that eighty percent of pulmonary fibrosis is idiopathic, that the remaining twenty percent is attributable to 160 different diagnoses, none of which is coal worker's pneumoconiosis, and that "pulmonary fibrosis has never been caused by simple coal worker's pneumoconiosis." *Id.* at 49.

Dr. Zaldivar is board-certified in internal medicine and pulmonary disease. He is also certified as a B-reader, and he had published two articles on blood gasses and pulmonary function in coal miners. *Id.* at 4-6.

Dr. Jarboe

Dr. Jarboe reviewed the medical records, including the examination reports from Drs. Rasmussen and Zaldivar, and concluded that the objective evidence is not sufficient to justify a diagnosis of coal worker's pneumoconiosis. EX 8 (August 12, 1998 report). He stated that there is not adequate radiographic evidence of pneumoconiosis, and although he disagreed with Dr. Zaldivar's assumption that pneumoconiosis only causes an obstructive impairment, he nonetheless concluded that the physiological evidence is not suggestive of dust-induced lung disease such as pneumoconiosis. He explained that epidemiological studies have shown that there is usually a concomitant reduction in both FVC and FEV_1 with coal dust exposure and that the preservation of FVC shown in Massie's ventilatory studies "argues strongly against the presence of a dust induced lung disease." *Id.* at 4. Dr. Jarboe also acknowledged that Massie showed a significant fall in oxygen tension with exercise, but he cited studies which reportedly showed that miners with simple coal worker's pneumoconiosis rarely, if ever, experienced physiological changes during exercise sufficient to cause a respiratory limitation of work capacity. Thus, he concluded that neither the radiographic nor physiological evidence supports a diagnosis of a dust-induced lung disease. *Id*.

Dr. Jarboe agreed that Massie has a respiratory or pulmonary impairment which is characterized by a significant drop in oxygen tension with exercise, and he stated that it is his opinion that Massie's cigarette smoking history, and not dust exposure, is the most likely cause of this impairment. *Id.* at 5. He further stated that he is "unable to totally explain the drop in oxygen tension which has occurred on exercise testing" which, he repeated, is "very unusual in coal miners unless they have advanced disease on x-ray." *Id.* He agreed with Dr. Zaldivar that Massie may have "a subtle interstitial fibrosis" which would need confirmation by high resolution CT scan, and he suggested that it is "remotely possible that he has emphysema which would account for a reduced diffusion capacity with fall in oxygen tension on exercise." *Id.* He continued, however, that he would attribute any emphysema in Massie's case to cigarette smoking rather than dust exposure, because "the degree of emphysema present in a miner's lung usually is proportionate to the amount of dust retention and fibrosis" and "[i]f there were enough

emphysema to cause a significant reduction in diffusion and fall in oxygen tension on exercise, I feel the x-ray would be abnormal." *Id.* Dr. Jarboe also concluded that Massie's disability is not caused, in whole or in part, by pneumoconiosis, stating, "[w]hile the cause of the fall in oxygen tension is not clear at this time, it is my reasoned opinion that there is no good evidence that it is due to coal dust exposure." *Id.* at 6.

Dr. Jarboe reiterated these opinions after reviewing some positive interpretations of chest x-rays and Dr. Forehand's report. EX 14 (September 29, 1998 Report). Dr. Jarboe is board-certified in internal medicine and pulmonary diseases, and he is a board-certified medical examiner and a certified B-reader. EX 8.

Dr. Fino

Dr. Fino reviewed the medical records and reported his findings to Consolidation's attorneys in a letter dated August 13, 1998. EX 8. He concluded that there is an interstitial pulmonary abnormality characterized by a decrease in pO2 with exercise. *Id.* at 6. He observed that this abnormality did not appear to have been present in 1994 when oxygen saturation tested as normal, and he stated that the significant drop in pO2 subsequently revealed by arterial blood gas studies in 1997 and 1998 showed development and progression of an interstitial pulmonary disease. *Id.* at 6-7. He further stated, "I would not expect coal workers' pneumoconiosis to present like this. I would have expected in 1994, the same year that he left the mines, to show a decrease in the oxygen saturation with exercise." *Id.* at 7.

Dr. Fino reviewed additional records and supplemented his initial findings in a report dated September 28, 1998. EX 14. Contrary to Dr. Jarboe, Dr. Fino stated that he agreed after reviewing a study from 1988 that coal miners can suffer an impairment in oxygen transfer. *Id.* at 3. However, he
stated that coal mine dust inhalation can not explain Massie's impairment in oxygen transfer in view of the fact that he had no such impairment when he was tested in 1994, the year he left coal mining: "The development of significant oxygen transfer abnormality within a couple of years is not consistent with a coal mine dust pulmonary condition." *Id.* at 4. Dr. Fino is a certified B-reader, and he is Board-certified in internal medicine with a subspecialty in pulmonary diseases. EX 8.

Dr. Loudon

Dr. Loudon reviewed the medical records and concluded that there in not sufficient objective evidence to justify a diagnosis of coal worker's pneumoconiosis. EX 8 (August 16, 1998 Report). He stated that he based his opinion on the "normal" chest x-rays and on the pulmonary and respiratory function test results. *Id.* at 5. While he found that Massie has a "significant respiratory impairment" in the form of gas exchange abnormalities, Dr. Loudon ruled out a diagnosis coal worker's pneumoconiosis:

> Abnormalities of the sort he has cannot, in my opinion, be attributed to coal worker's pneumoconiosis. Their cause or causes cannot, in my opinion, be determined from the records available, but they are likely to result from either an increase in the alveolar-arterial gradient for oxygen (and CO), or from ventilation-perfusion mismatch.

Id. In a supplemental report, Dr. Loudon reviewed additional medical evidence, including the examination report from Dr. Forehand. He stated that the report of an epidemiological study of West Virginia coal miners that was attached to Dr. Forehand's report[3] is relevant to Massie's case as it shows that heterogeneity of ventilation leading to arterial hypoxemia in a non-smoking miner can develop before changes appear on chest x-rays or in pulmonary function tests, but he stated that Massie's degree of hypoxemia is much more marked than that seen in the subjects of the study. EX 14 (September 27, 1998 Report) at 4. Dr. Loudon continued,

> Heterogeneity of ventilation, and ventilation/perfusion mismatch, are among the commonest causes of arterial hypoxemia, but they are seen a wide variety of conditions: pulmonary embolism, interstitial lung disease, chronic obstructive lung disease, asthma, granulomatous diseases, atelectasis; but these are often characterized by changes clinically, on x-ray, or on pulmonary function testing before they get to the point of causing hypoxemia.
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> I do not know why Mr. Massie has such marked arterial hypoxemia on exertion, but there are many possible causes. I was intrigued by the x-ray reports of eventration of the diaphragm; a high diaphragm could be associated with several mechanisms – such as eventration or phrenic nerve paralysis – that could cause exercise hypoxemia. Results of ventilation/perfusion scans would be helpful in seeking cause for hypoxemia.

Id. at 4-5. Dr. Loudon then concluded his supplemental report with the same opinions that he had set forth in his earlier report. The record shows that Dr. Loudon is a professor of medicine at the University of Cincinnati College of Medicine, and he is board-certified in internal medicine and pulmonary diseases. EX 8.

Dr. Castle

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[3] The study appended to Dr. Forehand's report is H. Susskind *et al.*, Heterogeneous Ventilation and Perfusion: A Sensitive Indicator of Lung Impairment in Non-smoking Coal Miners, 1 Eur Respir J 232-241 (1988). It is noted that Dr. Rasmussen is one of the authors of this study.

Dr. Castle reviewed the evidence of record for the Employer and concluded that Massie does not suffer from coal worker's pneumoconiosis. EX 10 at 7. He stated that Massie work in coal mining for a sufficient time, at least 30 years, to have developed coal worker's pneumoconiosis, and he stated that a second risk factor for the development of pulmonary disease is tobacco abuse, adding that 13 pack years of smoking is a "minimal recorded smoking history." *Id.* at 8. Dr. Castle found no radiographic evidence of coal worker's pneumoconiosis, and he stated that the physiologic studies are inconsistent with a diagnosis of coal worker's pneumoconiosis which, he said, causes a "mixed obstructive and restrictive ventilatory impairment." *Id.*[4] Dr. Castle further stated that it is very unusual for coal worker's pneumoconiosis to cause a reduction in diffusing capacity without radiographic evidence of either "p" or "r" type opacities, and he noted the studies cited by Dr. Jarboe as showing that coal worker's pneumoconiosis does not produce a drop in oxygen saturation with exercise. *Id.* He also indicated that he based his opinion that Massie's "arterial blood gas changes are not due to coal workers' pneumoconiosis" in part on his assumption that such changes would not be expected "in the absence of significant radiographic changes." *Id.* On the other hand, Dr. Castle stated that Massie's arterial blood gas impairment is "most likely representative of an interstitial pulmonary process unrelated to coal mining employment and coal dust exposure." *Id.* Thus, he concluded that "it is my opinion that Mr. Massie does not suffer from a chronic dust disease of the lungs that has been caused by, contributed to, or substantially aggravated by coal mine dust exposure." *Id*. at 8-9.

Dr. Castle reviewed additional records and submitted a supplemental report to Consolidation's attorneys. EX 14 (September 25, 1998 Report). His opinions did not change:

> Once again there was a significant fall in the pO_2 with exercise. For the reasons stated in my previous report, which included the lack of radiographic changes of pneumoconiosis by the majority of radiologists, the lack of physiologic changes of coal workers' pneumoconiosis, and the lack of any other findings indicative of coal workers' pneumoconiosis, other than the fall in pO_2, it is my opinion that a diagnosis of coal workers' pneumoconiosis cannot be made with respect to this gentleman.

Id. at 3-4. Dr. Castle had an opportunity to further explain his findings and opinions at a deposition taken on October 16, 1998. EX 16. He referred to the 1988 study of Welsh and West Virginian coal miners discussed by Dr. Rasmussen, and he stated that interstitial pulmonary fibrosis is part of the diagnosis of coal worker's pneumoconiosis. However, he disagreed with Dr. Rasmussen's position that the study demonstrates a greater incidence of interstitial pulmonary fibrosis among coal miners because the study's authors reported that there are no studies to show the overall prevalence of this condition in the general population. *Id.* at 11. Dr. Castle also

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[4] It is noted that Dr. Castle's description of the type of impairment caused by coal worker's pneumoconiosis is at odds with Dr. Zaldivar's opinion that the disease causes only an obstructive impairment.

acknowledged several other studies cited by Dr. Rasmussen but concluded that they did not really add anything to this particular case. *Id.* at 12-16.

Dr. Castle further testified that Massie has three types of respiratory abnormality – (1) a mild degree of airway obstruction, (2) a reduction in diffusing capacity and (3) abnormal gas exchange manifested by a fall in pO2 or hypoxemia with exercise. *Id.* at 17. His attention was drawn to the 1994 oximetry test results which he characterized as normal in contrast to the blood gasses in 1997 and 1998 which showed a significant drop in pO2 with exercise. *Id.* at 17-20. He stated that this finding is significant, and it led him to conclude that the abnormality in diffusion capacity and blood oxygenation with exercise occurred after 1994. *Id.* at 21. He also stated that Massie has a "clinically insignificant" degree of airway obstruction which was present in 1994, that this obstruction is "very likely" related to Massie's cigarette smoking and that it is possible that Massie could have developed a degree of emphysema which could be related to the obstruction and the reduction in diffusing capacity. *Id.* at 21-23. However, he further stated that it is possible that there is another cause, and he stated that it is his belief that Massie has developed some type of pulmonary interstitial process which has caused the reduction in diffusing capacity and drop in pO2 with exercise. *Id.* at 23. He explained that an interstitial pulmonary process may be present in the absence of a positive x-ray, much as one does not need a positive x-ray in order to have coal worker's pneumoconiosis. *Id.* at 25-26. He further explained that although fibrosis is a part of coal worker's pneumoconiosis, *idiopathic* pulmonary fibrosis is not associated with coal worker's pneumoconiosis. *Id.* at 26-27. He also contradicted Dr. Rasmussen's testimony that idiopathic pulmonary fibrosis is relatively rare, asserting that he sees the condition all the time in his pulmonary practice and that "diffuse interstitial lung disease probably are, after COPD, cancer and infections, are the next most common causes of lung disease that we see." *Id.* at 28.

On the question of the cause of Massie's pulmonary abnormalities, Dr. Castle testified that he does not believe that they are related to coal worker's pneumoconiosis which he described as an untreatable condition. *Id.* at 29. He said that it is entirely possible that Massie may have developed a degree of airway obstruction and a very mild degree of emphysema related to his smoking history, and he explained that although coal dust can cause evidence of *focal* emphysema, focal emphysema does not cause the type of abnormality seen in this case. *Id.* at 29-30. He further explained that with focal emphysema, one would expect to see "p" type opacities on a chest x-ray if focal emphysema was the cause of the diffusion capacity and gas exchange abnormalities. *Id.* at 30. He thus stated that it was his opinion that Massie does not have coal worker's pneumoconiosis as he found neither physical findings or radiographic evidence of coal worker's pneumoconiosis, and that Massie's recent onset of significant hypoxemia is related to the development of an underlying interstitial fibrosis. *Id.* at 31.

Dr. Castle additionally testified that he is familiar with the definition of legal pneumoconiosis and that he utilized that definition in formulating his opinions. Nevertheless, he stated that he did not believe that Massie suffers from legal pneumoconiosis:

> Mr. Massie, in my opinion, does not have anything that meets that criteria. I think he's disabled. I think he's disabled because of significant exercise-induced hypoxemia, but I am unable to render a diagnosis of pneumoconiosis for the reasons that I've stated. I think it's more likely that this man, with a negative x-ray, this man with basically a gas exchange abnormality, and the reduction in diffusing capacity that has occurred recently, most likely has developed another process which should be evaluated.

Id. at 32-33. Finally, Dr. Castle testified that the timing of the development of Massie's impairment (*i.e.*, rapidly between 1994 and 1997) supports a diagnosis of idiopathic pulmonary fibrosis rather than coal worker's pneumoconiosis because coal worker's pneumoconiosis may "stay the same forever" or "not get any worse" or "slowly progress" but not rapidly. *Id.* at 49-50.

Dr. Castle is the Co-Director of Respiratory Therapy Services at the Community Hospital of Roanoke Valley in Roanoke, Virginia. He is board-certified in internal medicine and pulmonary diseases, and he is a certified B-reader. He has also published several articles and given numerous presentations on diagnosis and treatment of lung disease. *Id.* at Deposition Exhibit 1; EX 10.

I have reconsidered these medical opinions as instructed by the Board. As an initial matter, I have reevaluated Dr. Rasmussen's opinion in light of the Board's finding that Drs. Jarboe and Castle both attributed Massie's mild obstructive impairment to cigarette smoking. While it is true that Dr. Rasmussen incorrectly relied on a negative smoking history when he examined Massie for the OWCP, I find that this discrepancy is of no import in assessing the credibility of any of the medical opinions in this record, including Dr. Rasmussen's. That is, there is a clear consensus of opinion that Massie's obstructive impairment is mild or, as Dr. Castle put it, clinically insignificant. Dr. Rasmussen addressed the discrepancy at his deposition and reasonably explained that the existence of a smoking history would not alter his opinions because he would expect to find more of an obstructive impairment if Massie's lung abnormalities were traceable to his past cigarette smoking. He did not directly contradict the opinions from Drs. Jarboe and Castle that the obstructive impairment is due to smoking; he simply concluded that it is not a determinative factor which is not inconsistent with any other physician's opinion. In my view, Massie's smoking history and the cause of his mild, clinically insignificant ventilatory obstruction is a red herring since no physician has attributed Massie's primary pulmonary abnormality (*i.e.*, the reduced diffusing capacity and drop in blood oxygenation with exercise) to cigarette smoking.

The real question to be addressed on remand is whether Dr. Rasmussen's finding that Massie suffers from legal pneumoconiosis is better reasoned and better supported by the objective medical evidence than the contrary medical opinions offered by Drs. Zaldivar, Jarboe, Fino, Loudon and Castle. There is no question regarding the existence of clinical or medical pneumoconiosis since the Board affirmed my rejection of Dr. Forehand's opinion and since Dr. Rasmussen and Consolidation's experts are in agreement that the evidence does not support a

diagnosis of clinical pneumoconiosis. I previously considered the relative credibility of the opinions from Drs. Rasmussen, Loudon and Castle regarding the existence of legal pneumoconiosis in my first decision on remand. I observed that "Dr. Loudon appears to have used the 'medical' or 'clinical' definition of pneumoconiosis rather than the substantially broader 'legal' definition" and I found it significant that "Dr. Loudon concede[d] that he was unable to identify the cause(s) of the Claimant's gas exchange abnormalities . . . and . . . stated that the type of abnormality seen in the Claimant is common to several conditions (*e.g.* COPD, interstitial lung disease and asthma) which can fall within the legal definition of pneumoconiosis if related to or aggravated by coal mine dust exposure." Decision and Order on Remand at 7-8. I further found that "while Dr. Loudon did suggest that the Claimant's gas exchange abnormalities are the likely result of either an increase in the alveolar-arterial gradient for oxygen and carbon monoxide or from a ventilation-perfusion mismatch, he did not state that these conditions are not related to or aggravated by the Claimant's extensive occupational exposure to coal mine dust." Decision and Order on Remand at 8. Based on these findings, I concluded that "Dr. Loudon's opinions carry little probative force in rebutting Dr. Rasmussen's well-reasoned diagnosis of legal pneumoconiosis." Decision and Order on Remand at 8. I similarly found that "Dr. Castle's conclusory dismissal of legal pneumoconiosis as a cause of the Claimant's pulmonary abnormalities is not sufficiently reasoned and supported by discussion of the objective evidence to constitute a reasoned medical opinion under section 718.202(a)(4) opposing a finding of legal pneumoconiosis." Decision and Order on Remand at 9. The Board did not specifically reject this weighing of the medical opinions, but it did express some concern as to whether I had made appropriate findings:

> Employer argues that the administrative law judge erred in according greater weight to the opinion of Dr. Rasmussen than to the contrary opinions of Drs. Castle and Loudon because Dr. Rasmussen considered the differences between legal pneumoconiosis and clinical pneumoconiosis. In finding Dr. Rasmussen's opinion well reasoned, the administrative law judge observed that "[u]nlike . . . several of the other physicians, Dr. Rasmussen clearly recognized the distinction between medical pneumoconiosis, a diagnosis which he would not make based on his assessment of the x-ray evidence, and legal pneumoconiosis." Decision and Order on Remand at 9. The administrative law judge stated, "I find that Dr. Castle's conclusory dismissal of legal pneumoconiosis as a cause of the [c]laimant's pulmonary abnormalities is not sufficiently reasoned and supported by discussion of the objective evidence to constitute a reasoned medical opinion under section 718.202(a)(4) opposing a finding of legal pneumoconiosis." *Id.* The administrative law judge also stated that "Dr. Loudon appears to have used the 'medical' or 'clinical' definition of pneumoconiosis rather than the substantially broader 'legal' definition of the pneumoconiosis which includes 'any pulmonary impairment related to or aggravated by dust exposure in the mines."' *Id.* at 7. Contrary to the administrative law judge's finding, the determination of whether a diagnosed condition is encompassed within the legal definition of pneumoconiosis

is for the fact-finder, and not for the physician. *See Hobbs v. Clinchfield Coal Co.*, 917 F.2d 790, 15 BLR 2-225 (4th Cir. 1990).

Slip opinion at 5-6 n.6. I agree with the Board's statement that it is the fact-finder's function to determine whether a diagnosed condition falls within the legal definition of pneumoconiosis, and I will endeavor in the following discussion to clearly elucidate my findings on this issue in the hope of avoiding further delay in the adjudication of this claim.

First of all, I find that Dr. Rasmussen's opinion that Massie's impaired respiratory function is due to his exposure to coal mine dust constitutes a diagnosis of a condition encompassed within the definition of legal pneumoconiosis. *See* 20 C.F.R. § 718.201(a)(2) ("'Legal pneumoconiosis' includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment."). I further find that Dr. Rasmussen's deposition testimony reflects that he exercised sound medical judgement in determining that Massie suffers from legal pneumoconiosis and that his diagnosis is based on objective medical evidence and supported by a reasoned medical opinion. Therefore, I conclude that Dr. Rasmussen's finding that Massie's respiratory impairment is caused by his exposure to coal mine dust supports a determination of the existence of pneumoconiosis pursuant to 20 C.F.R. § 718.202(a)(4).

Second, I find that Dr. Rasmussen's reasoned medical opinion that Massie suffers from pneumoconiosis is not outweighed by the contrary opinions from Drs. Zaldivar, Jarboe, Fino, Loudon and Castle, whether their opinions are considered individually or collectively. I reach this conclusion because I find that all of these physicians focused their inquiries almost exclusively on the question of whether there is sufficient evidence to justify a diagnosis of *clinical* pneumoconiosis. As the Fourth Circuit has instructed, this is no insignificant point, and an administrative law judge "must bear in mind when considering medical evidence that physicians generally use 'pneumoconiosis' as a *medical* term that comprises merely a small subset of the afflictions compensable under the Act." *Barber v. Director, OWCP*, 43 F.3d 899, 910 (4th Cir. 1995) (italics and internal quotation marks in original). While Consolidation's experts also attempted to rule out the existence of *legal* pneumoconiosis, I find for the reasons discussed below that their opinions on this issue are cursory, contradictory and, ultimately, less persuasive and well-reasoned than the diagnosis of legal pneumoconiosis rendered by Dr. Rasmussen.

I will begin with Dr. Zaldivar who attributed Massie's disabling pulmonary impairment to pulmonary fibrosis and stated that there are many causes of pulmonary fibrosis, but "coal worker's pneumoconiosis" is not one. EX 15 at 22, 49. Since it is clear from Dr. Zaldivar's testimony that he use the term "coal worker's pneumoconiosis" in the clinical or medical sense, I find that his statement, which appears to be contradicted by Dr. Castle's testimony that pulmonary fibrosis is a part of coal worker's pneumoconiosis (EX 14 at 26-27), only serves to negate clinical pneumoconiosis as a cause of Massie's pulmonary abnormality. It does not exclude Massie's coal mine dust exposure as a cause and, hence, does not equate to an opinion that Massie does not have legal pneumoconiosis. I further find Dr. Zaldivar's lengthy discussion of the rapid development of a gas exchange impairment after the normal test results in 1994 and his conclusion

that such rapid, post-exposure development is inconsistent with coal worker's pneumoconiosis is similarly limited to consideration of clinical pneumoconiosis which carries little weight in rebutting Dr. Rasmussen's diagnosis of legal pneumoconiosis. Lastly, I am reluctant to give much credence to Dr. Zaldivar's opinions on the cause of Massie's impairment in view of his testimony at the deposition where he mistakenly read the medical records as indicating that Massie is grossly obese and then proceeded with a spontaneous analysis that obesity is in part responsible for the impairment. In fairness to Dr. Zaldivar, this was an honest mistake which he readily acknowledged when Consolidation's attorney appropriately acted to correct the record. Though I find no evidence of medical sophistry, Dr. Zaldivar's *ad libitum* assessment nonetheless does erode my confidence that his opinions in this matter are the product of sound medical judgement.

Unlike Dr. Zaldivar, Dr. Jarboe used terminology such as "dust-induced lung disease" which suggests that he gave consideration to whether there is evidence that Massie suffers from legal pneumoconiosis. However, in concluding that the evidence does not support diagnosis of a dust-induced lung disease, I find that Dr. Jarboe effectively limited his analysis to clinical pneumoconiosis. Specifically, Dr. Jarboe concluded that Massie's drop in oxygen tension with exercise is more likely related to cigarette smoking than coal dust exposure because some studies have shown that miners with "simple coal worker's pneumoconiosis" rarely experience disabling physiological changes during exercise testing and because this type of abnormality is "very unusual in coal miners unless they have advanced disease on x-ray. EX 8 at 4-5. Thus, I find that Dr. Jarboe has attempted to rule out legal pneumoconiosis by reference to the characteristics of clinical pneumoconiosis. In my view, this is not sound reasoning, and I have given his opinion little weight in opposing Dr. Rasmussen's diagnosis. Moreover, I have credited the opinions from Dr. Rasmussen and Zaldivar that smoking is not a cause of Massie's gas exchange and diffusing capacity impairments because they both examined Massie and provided more detailed explanations for their dismissal of smoking as a causative factor. I also note that Dr. Jarboe's assumption that coal miners do not experience gas exchange impairments was directly refuted by Dr. Fino. EX 14 at 3.

Dr. Fino also used appropriate terminology in concluding that Massie's development of a significant oxygen transfer abnormality within a few years after his normal testing in 1994 is not consistent with a "coal mine dust pulmonary condition." EX 14 at 4. However, his opinions are cursory and lack the detail and substance underlying Dr. Rasmussen's contrary conclusions, and there is no evidence in the record that Dr. Fino has had anywhere near the extensive experience that Dr. Rasmussen has had in the study and diagnosis of occupational lung disease in coal miners. For these reasons, I have given his opinion less weight than Dr. Rasmussen's diagnosis of legal pneumoconiosis.

I find that Dr. Loudon's reports are clearly limited to consideration of whether the evidence supports a diagnosis of clinical pneumoconiosis. As I discussed in my first decision on remand, Dr. Loudon suggested that the Claimant's gas exchange abnormalities are the likely result of either an increase in the alveolar-arterial gradient for oxygen and carbon monoxide or from a ventilation-perfusion mismatch, but he did not state that these conditions are not related to or

aggravated by Massie's extensive occupational exposure to coal mine dust. Decision and Order on Remand at 8. Dr. Loudon also suggested that hypoxemia, as seen in this case, is characteristic of such conditions such as interstitial lung disease, chronic obstructive pulmonary disease and asthma, all of which fall within the definition of legal pneumoconiosis if related to coal mine dust exposure. *See Barber v. Director, OWCP*, 43 F.3d 899, 901 (4th Cir. 1995) (Act's broad definition of pneumoconiosis includes any pulmonary impairment related to or aggravated by dust exposure in the mines); *Richardson v. Director, OWCP*, 94 F.3d 164, 166, n.2 (4th Cir. 1996) (chronic obstructive pulmonary disease, if it arises out of coal mine employment, is clearly encompassed within the Act's definition of pneumoconiosis); *Hughes v. Clinchfield Coal Co.*, 21 BLR 1-134, 1-139 (1999) (chronic bronchitis and emphysema fall within the definition of pneumoconiosis if they are related to a claimant's coal mine employment). However, Dr. Loudon did not state that these possible diagnoses are unrelated to Massie's exposure to coal mine dust. I note that Dr. Loudon was also "intrigued" by the radiological findings of a diaphragm abnormality as a possible cause of hypoxemia, but I find his comments in this regard are closer to speculation than they are to a reasoned diagnosis. For these reasons, I find that Dr. Loudon has not made a medical finding that Massie does not suffer from legal pneumoconiosis and I again find that his opinions carry little probative force in rebutting Dr. Rasmussen's well-reasoned diagnosis of legal pneumoconiosis.

Finally, I find that Dr. Castle's opinions as to the presence of legal pneumoconiosis are flawed for the same reasons discussed with respect to the reports from Dr. Jarboe. That is, testimony was elicited from Dr. Castle that he understands the definition of legal pneumoconiosis and used the definition in formulating his opinions in this case. EX 16 at 32-33. However, much as Dr. Jarboe did, Dr. Castle attempted to dismiss legal pneumoconiosis by referring to the characteristics and findings associated with clinical pneumoconiosis. For example, in his first report, Dr. Castle concluded that Massie does not suffer from legal pneumoconiosis, EX 10 at 8-9, but did so after repeatedly noting the absence or radiographic evidence of clinical pneumoconiosis. *Id.* at 8. His second report continues the same narrow reference to clinical pneumoconiosis and its associated radiographic changes, EX 14, and these flaws were not cured at his deposition where Dr. Castle, despite his professed consideration of legal pneumoconiosis, consistently reverted to a narrow discussion of clinical pneumoconiosis. EX 16 at 29 ("coal worker's pneumoconiosis"), 29-30 ("focal emphysema" in which one expects to see "p" type opacities on x-ray), 32-33 (Massie does not meet the criteria for legal pneumoconiosis, in part, because of his "negative x-ray"), and 49-50 (the development of Massie's impairment is inconsistent with "coal worker's pneumoconiosis" which may either "not get any worse" or "slowly progress"). Incantation of the right words, in my view, does not amount to a reasoned diagnosis of no legal pneumoconiosis.

Based on the foregoing reconsideration in accordance with the Board's instructions, and giving greater weight to the diagnosis of legal pneumoconiosis by Dr. Rasmussen, I find that a preponderance of the medical opinion evidence establishes the existence of pneumoconiosis pursuant to 20 C.F.R.
§ 718.202(a)(4).

B. Weighing all Categories of Evidence Together

Since I have determined that medical opinion evidence establishes the existence of pneumoconiosis, I must next weigh together all categories of relevant evidence pursuant to *Island Creek Coal Co. v. Compton*, 211 F.3d 203, 210 (4th Cir. 2000) in determining whether the existence of pneumoconiosis has been established. I have previously concluded chest x-ray interpretations and medical opinions preponderate against a finding of clinical pneumoconiosis. There is no biopsy evidence to consider at 20 C.F.R. § 718.202(a)(2), and none of the presumptions described in 20 C.F.R. § 718.202(a)(3) are applicable. However, as the *Compton* court pointed out, "[e]vidence that does not establish medical pneumoconiosis, *e.g.*, an x-ray read as negative for coal workers' pneumoconiosis, should not necessarily be treated as evidence weighing *against* a finding of legal pneumoconiosis." *Compton*, 211 F.3d at 210 (italics in original). Although I find that the x-ray and medical opinion evidence is insufficient to establish that Massie suffers from clinical pneumoconiosis, I also find that this negative evidence does not carry much weight in terms of countering the medical opinion evidence establishing the presence of legal pneumoconiosis. That is, the two categories of evidence (x-rays and medical opinions on the presence of clinical pneumoconiosis versus the medical opinions regarding the existence of legal pneumoconiosis) address different questions. Therefore, I conclude, after weighing together all of the relevant evidence, that a preponderance of the evidence establishes that Massie suffers from pneumoconiosis as that term is defined in the Act and regulations.

C. Causal Relationship between Massie's Pneumoconiosis and Coal Mine Employment

The parties have stipulated that Massie has 23 years of qualifying coal mine employment. Therefore, Massie is entitled to use the rebuttable presumption at 20 C.F.R. § 718.203(b) to establish the causal relationship between his pneumoconiosis and coal mine employment. Based on my finding that the existence of legal pneumoconiosis has been established by a preponderance of the evidence, and noting that legal pneumoconiosis, by definition, connects a miner's condition with his coal mine employment, I find that the presumption is not rebutted. Accordingly, I conclude that Massie has met his burden of establishing that his pneumoconiosis arose out of his coal mine employment.

D. Disability Causation

A claimant must establish that pneumoconiosis is a substantially contributing cause of his total disability. 20 C.F.R. § 718.204(c). Pneumoconiosis is a substantially contributing cause if it "(i) Has a material adverse effect on the miner's respiratory or pulmonary condition; or (ii) Materially worsens a totally disabling respiratory or pulmonary impairment which is caused by a disease or exposure unrelated to coal mine employment." 20 C.F.R. §718.204(c)(1). In other words, pneumoconiosis must make more than a "negligible, inconsequential, or insignificant contribution" to the miner's total disability. 65 Fed.Reg. 79,920, 79,946 (December 20, 2000). Drs. Rasmussen, Forehand, Zaldivar, Jarboe, Fino, Loudon, and Castle all addressed the cause(s) of Massie's total disability. For the reasons discussed above in connection with my

reconsideration of the medical opinions bearing on the presence of pneumoconiosis, I credit Dr. Rasmussen's opinion that Massie's significant coal dust exposure is a major factor contributing to his totally disabling respiratory impairment over the contrary opinions of Drs. Zaldivar, Jarboe, Fino, Loudon, and Castle. As I noted in my first decision on remand, all of these physicians are all highly qualified specialists in pulmonary medicine. However, for the reasons discussed above, I find that their opinions on the cause of the Claimant's disabling pulmonary impairment are not as well-reasoned as the opinion offered by Dr. Rasmussen. Consequently, I conclude that a preponderance of the evidence establishes that Massie's pneumoconiosis is a substantially contributing factor to his total disability.

III. Conclusion

In sum, I have determined on remand that Massie has established the existence of pneumoconiosis arising out of coal mine employment and that he is totally disabled due to pneumoconiosis. As he has proven by a preponderance of the evidence that he is totally disabled due to pneumoconiosis, I conclude that Massie is entitled to an award of benefits to be paid by Consolidation. Since the date of onset of total disability due to pneumoconiosis can not be precisely ascertained on this record, benefits are payable from the first day of the month in which the current claim was filed. 20 C.F.R. §725.503(b); *Green v. Consolidation Coal Co.*, 790 F.2d 1118, 1119 n.4 (4th Cir. 1986). Benefits will be augmented on behalf of Massie's dependent spouse, Nancy Wriston Massie. 20 C.F.R. §§ 725.204, 725.205.

IV. Order

Consolidation Coal Company, as the responsible operator, shall pay to the Claimant Joseph E. Massie all benefits to which he is entitled under the Act, commencing July 1997 and continuing. Such benefits shall be augmented on behalf of the Claimant's dependent spouse, Nancy Wriston Massie.

SO ORDERED.

A

Daniel F. Sutton
Administrative Law Judge

Boston, Massachusetts
DFS:dmd

NOTICE OF APPEAL RIGHTS

Pursuant to 20 C.F.R. §725.481, any party dissatisfied with this Order may appeal it to the Benefits Review Board within thirty days from the date of this decision by filing a Notice of Appeal with the Benefits Review Board, ATTN: Clerk of the Board, P.O. Box 37601, Washington, DC 20013-7601. A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung

Benefits, Francis Perkins Building, Room N-2117, 200 Constitution Avenue, N.W., Washington, D.C. 20210.